

Research

Co-existence of panic disorder and sleep paralysis

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Sleep paralysis is a clinical phenomenon that is often a feature of narcolepsy. As its co-existence with panic disorder has not been noted before, we report on two cases in which the two conditions co-exist and postulate a neurochemical mechanism.

Key words: *sleep paralysis; narcolepsy; panic disorder; case report*

Introduction

The neurobiology of panic attacks has been the subject of much recent research that has led to the suggestion that central noradrenergic processes are important in this disorder (reviewed by Nutt and Lawson, 1992). Recently pharmacological studies have suggested that noradrenergic dysfunction also underlies certain features of narcolepsy, particularly the transient episodes of motor paralysis (cataplexy and sleep paralysis) (Nishino et al., 1990). This overlapping pathogenesis might lead one to expect that the two conditions might co-exist, although the only data on this point is the common experience that episodes of sleep paralysis often cause patients marked anxiety. The cases we report of two patients with panic disorder who also experience episodes of sleep paralysis may support the idea that the same central processes are involved in the two disorders.

Case reports

Case 1

The patient was a 26 year old male student of mixed Afro-Caribbean and Caucasian descent. His sleep paralysis started at the age of 16, when he had eaten some cheese and pineapple and had then developed a tingling feeling all over his body and had then felt unable to move anything apart from his eyes.

The episodes which had been wont to occur several times a year had become considerably more frequent in the 2 months prior to consultation; a period during which he had been taking larger than normal amounts of inhaled salbutamol. They occurred at - 2 a.m., were characterized by the same feelings as on his first attack at age 16 and lasted for a few seconds. He felt he was able to abort the episodes by trying hard to focus on something. Quite often the episodes were accompanied by sweating and afterwards he felt physically drained. They were made more likely by eating, drinking coffee or by having problems on his mind late at night. He found

these experiences profoundly distressing and was interested in finding a psychological explanation for them.

His panic attacks seem to have developed more insidiously. As a child he had been diagnosed as having asthma but he felt that the inhalers he was prescribed never really helped. Often when a doctor was called he recovered before he arrived making it quite likely that from an early age at least some of the 'asthma attacks' were, in fact, panic attacks. The patient agreed that this may have been the case and recognized that he was an anxious person who tended to worry a lot and who also felt mixed up and confused.

The diagnosis of panic disorder was made on the basis of, on average, two episodes of shortness of breath per day which occurred unexpectedly and were associated with a feeling of being smothered, nausea, increased heart rate, flushes and chills, depersonalization and chest pain and discomfort. These symptoms mostly occurred within the space of 10 min and were not caused by any organic factors. Attacks were more likely to occur if he was cold, if people were about and if he was wearing a personal stereo and was absorbed in that; despite this there was no real avoidance of these situations and he was able to carry on an active life as a youth worker and take on a social work diploma course. A possible reason for this was his rationalization of the attacks as asthma which responded almost immediately to salbutamol. During the period prior to his consultation he had been using his inhaler up to 15 times a day, but he had no evidence of chest pathology and his GP also felt that they were panic attacks.

Case 2

The patient was a 29 year old single, pregnant full time mother of four. Her sleep paralysis lasted for 1-1.5 min on each occasion. She described the sensation as 'my mind waking up when my body is still asleep'. The attacks had started about 5 years prior to presentation and she found the experience so unpleasant that she had started to drink on average eight units of alcohol per evening to relax her and forestall the attacks. When they first started they occurred once a night for 3 days running but at presentation were occurring once every 2 months.

Her complaint was, in fact, of panic attacks which had started 5 months previously, at the beginning of her pregnancy. These seemed to have developed on the back-ground of increasing anxiety which had started at the time of her third pregnancy when she was still married to her first husband and the relationship had been difficult. The panic attacks started at a time when she was drinking heavily and her (second) husband was unhappy about her doing this whilst she was pregnant. The diagnosis of panic disorder was made on the basis of the attacks being unexpected and occurring at least four times in 4 weeks. She experienced a fear of dying, breathlessness, sweating and palpitations. These all occurred within 10 min of each other and were not caused by organic factors. The attacks could occur at any time and in any place and were accompanied by subjective anxiety but she was not agoraphobic.

Discussion

Current theories of noradrenergic involvement in panic disorder suggest that this system is overly labile, being hyper-responsive to both activating and inhibiting

interventions (see Nutt and Lawson, 1992). Similarly, episodic paralysis of the sort occurring during REM sleep and in narcolepsy is thought to be due to a transient inhibition of noradrenergic outflow. In narcoleptic dogs the lesion has been identified as a doubling of the number of inhibitory α_2 -adrenoceptors in the locus coeruleus (Fruhstorfer et al., 1989). This results in bouts of excessive inhibition which are manifested as episodes of REM sleep and motor paralysis. Studies of CSF in man and dogs find evidence of reduced noradrenaline in those with narcolepsy.

In patients with panic disorder there is clinical evidence for increased pre-synaptic inhibitory α_2 -adrenoceptor sensitivity (Charney and Heninger, 1986; Nutt, 1989) which could lead to transient reductions in outflow similar to those seen in narcolepsy. It is possible that these could explain certain features of panic attacks such as the muscle weakness and fainting.

A further point of similarity between some symptoms of narcolepsy and panic disorder is the fact that both conditions respond to treatment with tricyclic antidepressants and MAOIs (Lishman, 1987; Lydiard, 1987; Nutt and Glue, 1989; Rifkin et al., 1981), perhaps because they down-regulate pre-synaptic α_2 -adrenoceptors (Charney, Menkes and Heninger, 1981).

These interesting parallels between panic disorder and narcolepsy may be worth further exploration. Although our patients do not meet the criteria for the diagnosis of narcolepsy, it is possible that they may later develop it. Thus it will be interesting to monitor the evolution of these two conditions particularly in response to treatment.

Note added in proof. We have since discovered a report on association of sleep paralysis and panic disorder in African-Americans (Bell et al., 1986. *J. Natl. Med. Assoc.* 75: 649-659).

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